



# Application of control and optimal treatment for predator-prey model<sup>†</sup>

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## Abstract

Mathematical ecology and mathematical epidemiology are major fields in both biology and applied mathematics. In the present paper, a four-dimensional eco-epidemiological model with infection in both prey and predator populations is studied. It consists of susceptible prey, infected prey, susceptible predator, and infected predator. The functional response is assumed to be of Lotka–Volterra type. The behavior of the system such as the existence, boundedness, and stability for solutions and equilibria are studied and also the basic reproduction number for the proposed model is computed. Moreover, a related control model and optimal treatment for the control model are presented. Finally, to verify the analytical discussion, a numerical simulation is carried out.

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## 1 Introduction

Health is the most important topic in the world for human, animal, plant, and any animate entity. Anybody gets involved with many various viruses such

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as Black Death (1300), Spanish Flu (1918), HIV/AIDS (1920), Swine Flu (2009), Ebola virus (2013), Zika virus (2016), Corona viruses such as: SARS-Cov(2003), MERS-Cov(2012), COVID-19 (2019), and so on, which are the most deadly and disastrous viruses in 20th and 21st centuries. Some of them are not only epidemic but also are pandemic (that is a world epidemic). Black Death is just the most famous epidemic historically. The history of epidemic is an ever fascinating area. Hence nowadays, the study of diseases and their transmission is most studied subject. Therefore, physicians have to apply mathematician knowledge to protect man against infected diseases transmitted by viruses. It is clear that ecology, biology, and epidemiology are major areas. Therefore, many researchers join together to work on common topics and interdisciplinary subjects. The above sentences motivate mathematicians, physicians, researchers, and scientists to join together in the said areas such as dynamics of infectious disease by the help of theory of modeling, dynamical systems, and so on.

Infectious diseases may be a major factor in human population size. One of them is Black Death in the 14th century, which killed up to one-fourth of the people (which occurred in Eurasia); see [6]. Diseases of European people such as smallpox that were carried out by Cortez and coworkers to Mexico, decimated native population there in the 16th century. Rinderpest caused high mortality in wild animals in Africa at the end of the 19th century.

It is obvious that describing any type of ecological phenomena such as predator-prey, coexistence, populations reactions, and the like entails having sufficient mathematical knowledge. Mathematical models may be applied as an effective tool to analyze such realistic phenomena. In the complex ecosystem some reactions such as coexistence, competition, food chain, food web, and predator-prey relationships may be important in regulating numbers of population of species. Whenever a bounty was placed on natural predators in the Kaibab Plateau in Arizona, the number of deer population went up for food supply, and then over half of the deer died in 1923–25; see [6]. Regarding to new researches, we may see an increase in a number of works that describe the relationships between demographic process among different populations and disease. Mathematical biologists indeed have been working on merging ecology and epidemiology (see [2, 7, 8, 5, 10, 12, 14, 15, 16]).

This research work describes a predator and prey type by using biological and mathematical modeling. This model consists of two species: prey species and the predator species. Any species has two subclasses: susceptible and infected.

The original parts of the paper are constructed as follows: Section 2 consists the mathematical modeling; indeed a model is formulated. Section 3 involves stability analysis consisting boundedness, equilibria, stability, and the basic reproduction number  $R_0$  for model (2). In section 4, we analyze a control model having infection. Also an optimal treatment approach is studied. Indeed, this section consists making control model for system (2), applying the control functions for system (3) and its analysis, and establishing

an optimal control to minimize the number of infected species. In section 5, to verify the analytical discussion, numerical simulations are computed. Finally, in the last section conclusion is presented.

## 2 Mathematical modeling

In this section, a predator-prey system having infection is described and then its mathematical model is presented; see [1]. First assume that  $S(T)$ ,  $I(T)$ ,  $X(T)$ , and  $Y(T)$  describe the densities of susceptible prey, infected prey, susceptible predator, and infected predator at time  $T$ , respectively. In the second step, some basic hypotheses which have been made in the formulating of model, are presented as follows:

- $H_1$ ) The prey species grow logistically having intrinsic growth rate  $\gamma$  and carrying capacity  $K$  (which is positive) in the case when there is no diseases and predator species.
- $H_2$ ) Just susceptible prey may reproduce.
- $H_3$ ) Prey species can be infected never recovers, which will either die or be removed in predation interacting. The infected prey species have a disease induced death.
- $H_4$ ) Infected predators species can catch a healthy predator. Meanwhile, infected prey may be available for predation by predators.
- $H_5$ ) By considering a direct contact with infected predator, disease may be spread.
- $H_6$ ) Finally, consider predator species die naturally, but infected species have a disease induce excess death rate also.

These mentioned considerations motivate one to the following system:

$$\begin{aligned} \frac{dS}{dT} &= \gamma S \left(1 - \frac{S+I}{K}\right) - a_1 SI - b_1 SX, \\ \frac{dI}{dT} &= a_1 SI - d_1 I - f_1 IX - m_1 IY, \\ \frac{dX}{dT} &= c_1 SX + g_1 IX - e_1 XY - \delta_1 X, \\ \frac{dY}{dT} &= e_1 XY - (\delta_1 + \alpha_1)Y + n_1 IY, \end{aligned} \quad (1)$$

with the following initial values  $S(0) = S_0$ ,  $I(0) = I_0$ ,  $X(0) = X_0$ ,  $Y(0) = Y_0$ . We moreover make an obvious assumption that all the parameters are positive. Indeed, the parameters are described as follows:

- $i$ )  $a_1$  and  $b_1$  are infection rates for prey population and predation rate of susceptible prey by healthy predators.
- $ii$ )  $d_1$  is an infected prey species disease induced death.

- iii)  $f_1$  and  $m_1$  describe predation rate of infected prey by susceptible and infected predators, respectively.
- iv)  $c_1$  and  $g_1$  describe conversion rates for healthy and infected preys to healthy predator, respectively.
- v)  $\delta_1$  describes the natural death rate for the predator population having a disease induced death rate  $\alpha_1$ .
- vi)  $e_1$  and  $n_1$  denote infection rates for predator population and conversion rate for infected prey to infected predator.

By using the following scaling

$s = \frac{S}{K}$ ,  $i = \frac{I}{K}$ ,  $x = \frac{X}{K}$ ,  $y = \frac{Y}{K}$ , and  $t = \gamma T$ , after some simplification, system (1) reads as follows:

$$\begin{aligned} \frac{ds}{dt} &= s(1 - s - i) - asi - bsx, \\ \frac{di}{dt} &= asi - di - fxi - miy, \\ \frac{dx}{dt} &= csx + gix - exy - \delta x, \\ \frac{dy}{dt} &= exy - \alpha y + niy. \end{aligned} \tag{2}$$

In the above system, we have

$$\begin{aligned} a &= \frac{a_1 K}{\gamma}, & b &= \frac{b_1 K}{\gamma}, & c &= \frac{c_1 K}{\gamma}, & d &= \frac{d_1}{K}, & e &= \frac{e_1 K}{\gamma}, & f &= \frac{f_1 K}{\gamma}, \\ g &= \frac{g_1 K}{\gamma}, & m &= \frac{m_1 K}{\gamma}, & n &= \frac{n_1 K}{\gamma}, & \alpha &= \frac{\delta_1 + \alpha_1}{\gamma}, & \delta &= \frac{\delta_1}{\gamma}. \end{aligned}$$

We are now going to study the solutions boundedness for system (2). For system (2), any solution that starts in  $R_+^4$ , remains in this area.

As regarding any parameters nonnegative, one is able to see that the right-side of model (2) can be a smooth function at  $(s, i, x, y)$  in the first octant,

$$\Omega = \{(s, i, x, y) | s \geq 0, i \geq 0, x \geq 0, y \geq 0\}.$$

Also  $\Omega$  is an invariant set.

Since system (2) is homogeneous, we have  $s = 0, i = 0, x = 0$ , and  $y = 0$  is the trivial solution. The uniqueness and existence theorem ensures that any trajectory starting from the first quadrant remains in it, that is, no trajectory will cross the coordinate planes.

### 3 Stability analysis

In the present section, the solution behavior such as boundedness and stability for equilibria, is studied. Then, the basic reproduction number for the said model is computed.

### 3.1 Equilibria Stability

**Theorem 1.** [1] Any solution for system (2) is uniformly bounded.

In the present section, first the equilibrium points and also the basic reproduction number  $R_0$  are computed for system (2). Then the stability analysis for these equilibria will be presented. Now, we are going to find out equilibria for system (2) as follows:

a)  $E_0(0, 0, 0, 0)$ , which is known as the trivial equilibrium.

b)  $E_1(1, 0, 0, 0)$ , which is known as the axial equilibrium.

c)  $E_2(s_2, 0, x_2, 0)$ , which is known as the disease-free equilibria. In the truth of the following conditions, this equilibrium exists:  $c > \delta$ ,  $s_2 = \frac{\delta}{c}$ , and  $x_2 = \frac{c-\delta}{bc}$ . Moreover, this condition  $c > \delta$  indicates that  $c_1 > \frac{\delta_1}{K}$ , that is, a disease-free equilibrium exists provided that predator is a high capacity consumer.

d)  $E_3(s_3, i_3, 0, 0)$ , which is known as the predator-free equilibria;

In the truth of the following conditions, this equilibrium exists  $a > d$ ,  $s_3 = \frac{d}{a}$ ,  $i_3 = \frac{a-d}{a(1+a)}$ . Furthermore, this condition  $a > d$  implies  $a_1 K > d_1$ , that is, it indicates that the predator-free equilibrium exists provided that a disease induced death rate is low enough.

e)  $E_4(s_4, i_4, x_4, 0)$ , which is known as the infected-predator-free equilibria. This exists when  $s_4, i_4, x_4$  are given by

$$\begin{aligned} s_4 &= \frac{g(bd + f) - \delta(f + af)}{g(ab + f) - c(f + af)}, \\ i_4 &= \frac{\delta(ab + f) - c(bd + f)}{g(ab + f) - c(f + af)}, \\ x_4 &= \frac{ag(bd + f) - gd(ab + f) + (f + af)(cd - a\delta)}{gf(ab + f) - cf(f + af)}. \end{aligned}$$

f)  $E_5(s_5, 0, x_5, y_5)$ , which is known as the infected-prey-free equilibria.

In the case of  $e > b\alpha$  and  $ce > bc\alpha + e\delta$ , this equilibrium exists, where  $s_5, x_5, y_5$  are given as

$$\begin{aligned} s_5 &= \frac{e - b\alpha}{e}, \\ x_5 &= \frac{\alpha}{e}, \\ y_5 &= \frac{ce - bc\alpha - e\delta}{e^2}. \end{aligned}$$

g)  $E^*(s^*, i^*, x^*, y^*)$ , which is known as the interior equilibrium point; it exists provided that

$$(i) |P_2| \geq \max\{|(1+a)P_1 + bP_3|, \left|\frac{(c+ac-g)P_1+bcP_3}{c-\delta}\right|\},$$

(ii)  $P_1, P_2$ , and  $P_3$  are of the same sign,

where  $P_1, P_2$ , and  $P_3$  are given by

$$\begin{aligned} P_1 &= e(ab\alpha + f\alpha + cm + de) - (bcm\alpha + me\delta + ae^2), \\ P_2 &= e(abn + fn + (1+a)cm) - (bcmn + a(1+a)e^2 + meg), \\ P_3 &= (aen + mn\delta + (1+a)cm\alpha) - cmn + den + (1+a)ae\alpha + mg\alpha. \end{aligned}$$

If the above conditions are true, then the values of  $s^*, i^*, x^*, y^*$  may be found by

$$\begin{aligned} s^* &= \frac{P_2 - (1+a)P_1 - bP_3}{P_2}, \\ i^* &= \frac{P_1}{P_2}, \\ x^* &= \frac{P_3}{P_2}, \\ y^* &= \frac{(g - c - ac)P_1 + (c - \delta)P_2 - bcP_3}{eP_2}. \end{aligned}$$

Now we are able to analyze the stability of the above equilibrium points for system (2). Indeed, some conditions are presented which show the points  $E_1, E_2, E_3, E_4, E_5$  and  $E^*$  are locally asymptotically stable.

**Theorem 2.** [1] The following statements are true for system (2).

- (a) The trivial equilibrium  $E_0$  is unstable.
- (b) If  $d > a$  and  $\delta > c$ , then the equilibrium  $E_1$  is locally asymptotically stable.
- (c) If  $e\delta + b\alpha > ec$ ,  $A_1 > 0$ ,  $A_3 > 0$ , and  $A_1 A_2 > A_3$ , then the equilibrium  $E_2$  is locally asymptotically stable.
- (d) If  $n(a - d) < a\alpha(1 + a)$  and  $g(a - d) + (cd - a\delta)(1 + a) < 0$ , then the equilibrium  $E_3$  is locally asymptotically stable.
- (e) If  $c < \delta$ ,  $B_1 > 0$ ,  $B_3 > 0$ , and  $B_1 B_2 - B_3 > 0$ , then the equilibrium  $E_4$  is locally asymptotically stable.
- (f) If  $C_1 > 0$ ,  $C_3 > 0$ ,  $C_4 > 0$  and  $C_1 C_2 C_3 > C_3^2 + C_1^2 C_4$ , then the equilibrium  $E_5$  is locally asymptotically stable.
- (g) If  $D_1 > 0$ ,  $D_3 > 0$ ,  $D_4 > 0$  and  $D_1 D_2 D_3 > D_3^2 + D_1^2 D_4$ , then the equilibrium  $E^*$  is locally asymptotically stable.

### 3.2 The basic reproduction number

The next generation matrix method [3] is used to calculate the basic reproduction number  $R_0$ ; see [9]. Clearly,  $i$  and  $y$  are the relevant classes of infection. The classes  $i(t), y(t)$  from our model are

$$\begin{aligned}\frac{di}{dt} &= asi - di - fxi - miy, \\ \frac{dy}{dt} &= exy - \alpha y + niy.\end{aligned}$$

To define the basic reproduction number, denote the vectors  $\varphi$  and  $\nu$  and the inflow and outflow from the disease compartment  $i$  and  $y$  as follows:

$$\begin{aligned}\varphi &= \begin{pmatrix} \varphi_1 \\ \varphi_2 \end{pmatrix} = \begin{pmatrix} asi \\ exy + niy \end{pmatrix}, \\ \nu &= \begin{pmatrix} \nu_1 \\ \nu_2 \end{pmatrix} = \begin{pmatrix} di + fxi + myi \\ \alpha y \end{pmatrix}.\end{aligned}$$

We compute the  $2 \times 2$  Jacobian matrices, evaluated at the disease-free equilibrium point  $E_2(s_2, 0, x_2, 0)$ ,

$$F = \left( \frac{\partial \varphi_i}{\partial x_j}(s_2, 0, x_2, 0) \right)$$

and

$$V = \left( \frac{\partial \nu_i}{\partial x_j}(s_2, 0, x_2, 0) \right)$$

Therefore, two matrices  $F$  and  $V$  corresponding to the gain and loss components of system (2) are defined as

$$F = \begin{pmatrix} as & 0 \\ ny & ex + ni \end{pmatrix}_{|E_2} = \begin{pmatrix} as_2 & 0 \\ 0 & ex_2 \end{pmatrix}$$

and

$$V = \begin{pmatrix} d + fx + my & mi \\ 0 & \alpha \end{pmatrix}_{|E_2} = \begin{pmatrix} d + fx_2 & 0 \\ 0 & \alpha \end{pmatrix}.$$

Now, the next generation matrix is defined as

$$G = FV^{-1} = \begin{pmatrix} \frac{as_2}{d+fx_2} & 0 \\ 0 & \frac{ex_2}{\alpha} \end{pmatrix},$$

$$\lambda^2 - \frac{(ex_2d + ex_2^2f + as_2\alpha)}{\alpha(d + fx_2)}\lambda + \frac{ex_2as_2}{\alpha(d + fx_2)} = 0.$$

The basic reproduction number indeed may be dominant eigenvalue for the next generation matrix. Thus,

$$R_0 = \frac{1}{2} \left( \frac{(ex_2d + ex_2^2f + as_2\alpha)}{\alpha(d + fx_2)} + \sqrt{\frac{(\alpha as_2)^2 + (ex_2)^2(d + fx_2)^2}{\alpha^2(d + fx_2)^2} - 4 \frac{ex_2as_2}{\alpha(d + fx_2)}} \right),$$

where

$$s_2 = \frac{\delta}{c}, \quad x_2 = \frac{c - \delta}{bc}.$$

If  $R_0 > 1$ , then the disease is endemic. If  $R_0 < 1$ , then the disease-free equilibrium is locally asymptotically stable.

#### 4 Control model and optimal treatment

In this section, first we make a control model for system (2) and then analyze the application of control for system (3). After it we describe an optimal control approach.

By this mean, we add the control functions  $u_1$  and  $u_2$  as follows:

- (i)  $u_1$  is denoted as the control factor for the first and second species (prey and infected prey species).
- (ii)  $u_2$  is denoted as the control factor for the third and fourth species (predator and infected predator species).

The role of control functions is to treat the infected prey and predator species. Hence, the control system can be written as follows:

$$\begin{aligned} \frac{ds}{dt} &= s(1 - s - i) - a(1 - u_1)si - bsx, \\ \frac{di}{dt} &= a(1 - u_1)si - di - fxi - miy, \\ \frac{dx}{dt} &= csx + gix - e(1 - u_2)xy - \delta x, \\ \frac{dy}{dt} &= e(1 - u_2)xy - \alpha y + niy, \end{aligned} \tag{3}$$

where  $0 \leq u_i \leq 1, i = 1, 2$ . When  $u_i = 0$ , no treatment occurs and whenever  $u_i = 1$ , the model shows the full treatment. To investigate the model, among all of equilibrium points, we analyze the interior equilibrium, that is,  $E^*(s^*, i^*, x^*, y^*)$  for system (3). It is easy to show that  $E^*$  exists, if

- (i)  $|P_2| \geq \max\{|(1 + a(1 - u_1))P_1 + bP_3|, |\frac{(c + a(1 - u_1)c - g)P_1 + bcP_3}{c - \delta}| \}$ ,
- (ii)  $P_1, P_2$ , and  $P_3$  are of the same sign, where  $P_1, P_2$ , and  $P_3$  are given by

$$\begin{aligned}
P_1 &= e(1-u_2)(a(1-u_1)b\alpha + f\alpha + cm + de(1-u_2)) \\
&\quad - (bcm\alpha + me(1-u_2)\delta + a(1-u_1)e^2(1-u_2)^2), \\
P_2 &= e(1-u_2)(a(1-u_1)bn + fn + (1+a(1-u_1)cm) \\
&\quad - (bcmn + a(1-u_1)(1+a(1-u_1))e^2(1-u_2)^2 + me(1-u_2)g), \\
P_3 &= (a(1-u_1)e(1-u_2)n + mn\delta + (1+a(1-u_1))cm\alpha) - cmn + de(1-u_2) \\
&\quad + (1+a(1-u_1))a(1-u_1)e(1-u_2)\alpha + mga\alpha.
\end{aligned}$$

When the above equalities are satisfied, the values of  $s^*, i^*, x^*, y^*$  are as follows:

$$\begin{aligned}
s^* &= \frac{P_2 - (1+a(1-u_1))P_1 - P_3b}{P_2}, \\
i^* &= \frac{P_1}{P_2}, \\
x^* &= \frac{P_3}{P_2}, \\
y^* &= \frac{(g - c - a(1-u_1)c)P_1 + (c - \delta)P_2 - bcP_3}{e(1-u_2)P_2}.
\end{aligned}$$

**Theorem 3.** The nontrivial equilibrium point  $E^*$  is locally asymptotically stable for model (3) provided  $D'_1 > 0, D'_3 > 0, D'_4 > 0$  and  $D'_1 D'_2 D'_3 > D'^2_3 + D'^2_1 D'_4$ .

*Proof.* At the interior equilibrium  $E^*$ , the Jacobian matrix  $J(E^*)$  can be obtained as follows:

$$J(E^*) = \begin{pmatrix} -s^* & -(1+a(1-u_1))s^* & -bs^* & 0 \\ a(1-u_1)i^* & 0 & -fx^* & -mi^* \\ cx^* & gx^* & 0 & -e(1-u_2)x^* \\ 0 & ny^* & e(1-u_2)y^* & 0 \end{pmatrix}$$

The corresponding characteristic equation is given by

$$\lambda^4 + D'_1\lambda^3 + D'_2\lambda^2 + D'_3\lambda + D'_4 = 0,$$

where

$$\begin{aligned}
D'_1 &= s^*, \\
D'_2 &= e^2(1-u_2)^2x^*y^* + fgx^{*2} + a(1-u_1)[a(1-u_1) + 1]s^*i^* + mni^*y^* \\
&\quad + bcs^*x^*, \\
D'_3 &= e^2(1-u_2)^2s^*x^*y^* + fgs^*x^{*2} + mns^*i^*y^* - e(1-u_2)nf x^{*2}y^* \\
&\quad + me(1-u_2)gi^*x^*y^* + a(1-u_1)bgs^*i^*x^* - cf(a(1-u_1) + 1)s^*x^*,
\end{aligned}$$

and

$$\begin{aligned}
D'_4 = & -e(1-u_2)nfs^*x^{*2}y^* + [e(1-u_2)mg \\
& + a(1-u_1)(a(1-u_1)+1)e(1-u_2) - a(1-u_1)be(1-u_2)n \\
& + bcmn - cme(1-u_2)[a(1-u_1)+1]]s^*i^*x^*y^*.
\end{aligned}$$

Regarding the Routh–Hurwitz criterion, it is easy to see that if

$$\begin{cases} D'_i > 0, & i = 1, 3, 4, \\ D'_1(D'_2D'_3 - D'_1D'_4) - D'^2_3 > 0, \end{cases}$$

then all of the eigenvalues for  $J(E^*)$  have negative real parts.

Therefore, if  $D'_1 > 0$ ,  $D'_3 > 0$ ,  $D'_4 > 0$  and  $D'_1D'_2D'_3 > D'^2_3 + D'^2_1D'_4$ , then  $E^*$  is locally asymptotically stable.  $\square$

Now, we describe an optimal control approach. As it is well known, the optimal control problem consists of forcing the solution for a system out of undesirable set at a given time. We are going to control the population of infected prey and predators, in an optimal method. And so, we establish an optimal criterion that consists of the minimizing of total number for infected species along with the treatment costs. We need to find the control functions  $u_1, u_2$ , as defined before, such that minimize the objective functional

$$\min \int_0^T \left[ \frac{1}{2}(W_1u_1^2 + W_2u_2^2) + i(t) + y(t) \right] dt. \quad (4)$$

The existence of an optimal control for the state system is analyzed by using the theory developed by Fleming et al. [?]. The boundedness of the solutions was discussed in section 3, which is needed to obtain the existence of an optimal control. Here, we may state the existence theorem as follows:

**Theorem 4.** There exist optimal controls  $u_1$  and  $u_2$  that minimize the objective functional (4), if the following conditions are met:

1. The class of all initial conditions with controls  $u$  and  $v$  such that  $u$  and  $v$  are Lebesgue integrable functions on  $[0, T]$  with values in the admissible control set along with each state equation being satisfied is not empty.
2. The admissible control set is closed and convex.
3. The right-hand side of the state system is continuous, bounded above by a sum of the bounded control and the state, and it can be written as a linear function of  $u_1$  and  $u_2$  with coefficients depending on time and the state variables.
4. The integrand of the functional is convex on the admissible control set and is bounded above.

The classical approach is described in [11], so we can derive the associated Hamiltonian as:

$$\begin{aligned} H = & \frac{1}{2} (W_1 u_1^2 + W_2 u_2^2) + i(t) + y(t) + p_s(t)(s(1 - s - i) - a(1 - u_1)si - bsx) \\ & + p_i(t)(a(1 - u_1)si - di - fxi - miy \\ & + p_x(t)(csx + gix - e(1 - u_2)xy - \delta x) \\ & + p_y(t)(e(1 - u_2)xy - \alpha y + niy). \end{aligned}$$

where the functions  $p_s(t), p_i(t), p_x(t), p_y(t)$  are called as the co-state variables.

These variables should satisfy in the following equations:

$$\frac{dp_s(t)}{dt} = -\frac{\partial H}{\partial s}, \quad \frac{dp_i(t)}{dt} = -\frac{\partial H}{\partial i}, \quad \frac{dp_x(t)}{dt} = -\frac{\partial H}{\partial x}, \quad \frac{dp_y(t)}{dt} = -\frac{\partial H}{\partial y},$$

or explicitly

$$\begin{aligned} \frac{dp_s(t)}{dt} &= -\frac{\partial H}{\partial s} \\ &= -(p_s(t) - 2sp_s(t) - ip_s(t) - aisp_s(t) + aisu_1p_s(t) - bxp_s(t)), \\ \frac{dp_i(t)}{dt} &= -\frac{\partial H}{\partial i} = -(1 - sp_s(t) - asp_s(t) + asu_1p_s(t) + asp_i(t) \\ &\quad - asu_1p_i(t) - dp_i(t) - fxi p_i(t) - mip_i(t) + gxp_x(t) + nyp_y(t)), \\ \frac{dp_x(t)}{dt} &= -\frac{\partial H}{\partial x} = -(-bsp_s(t) - fip_i(t) + csp_x(t) + gip_x(t) - eyp_x(t) \\ &\quad + eyu_2p_x(t) - \delta p_x(t) + eyp_y(t) - u_2eyp_y(t)), \\ \frac{dp_y(t)}{dt} &= -\frac{\partial H}{\partial y} = -(1 - mip_i(t) - exp_x(t) + exu_2p_x(t) + exp_y(t) \\ &\quad - exu_2p_y(t) - \alpha p_y(t) + nip_y(t)). \end{aligned} \tag{5}$$

For the above system of co-stated variables (5), we consider the following boundary conditions:

$$s(0) = s_0, \quad i(0) = i_0, \quad x(0) = x_0, \quad y(0) = y_0,$$

and

$$p_s(T) = p_i(T) = p_x(T) = p_y(T) = 0.$$

Differentiating  $H$  with respect to  $u_1$  and  $u_2$  yields:

$$\begin{aligned} \frac{\partial H}{\partial u_1} &= W_1 u_1 + asi(p_s(t) - p_i(t)) = W_1 u_1 + (asi)p_s(t) - (asi)p_i(t) = 0, \\ \frac{\partial H}{\partial u_2} &= W_2 u_2 + exi(p_x(t) - p_y(t)) = W_2 u_2 + (exi)p_x(t) - (exi)p_y(t) = 0. \end{aligned}$$

Thus, the control factors  $u_1$  and  $u_2$  can be obtained as follows:

$$u_1(t) = \frac{asi}{W_1}(-p_s(t) + p_i(t)),$$

$$u_2(t) = \frac{exi}{W_2}(-p_x(t) + p_y(t)).$$

As regards  $0 \leq u_1, u_2 \leq 1$ , we have the optimal control laws as

$$u_1^*(t) = \min\{1, \max\{0, u_1\}\} = \min\{1, \max\{0, \frac{asi}{W_1}(p_i(t) - p_s(t))\}\},$$

$$u_2^*(t) = \min\{1, \max\{0, u_2\}\} = \min\{1, \max\{0, \frac{exi}{W_2}(p_y(t) - p_x(t))\}\}.$$

## 5 Discussion and numerical simulation

It is obviously that the numerical simulation is important beside the analytical findings to verify them. By using MATLAB software, we now present computer simulation for different solutions of systems (2) and (3). We first present a computer simulation for equilibrium of system (2). The behavior of these equilibria are shown in Figures 1–6. By setting the parameters and initial conditions for system (2), we can construct Tables 1 and 2, which are referred in Figures 1–6. These values are calculated in MATLAB software.

Now, we are going to illustrate Figures 1–6. First, we take the parameters and initial condition for system (2) as Row No. *i* of Tables 1 and 2. Then we see that the conditions of Theorem 2 are satisfied and consequently  $E_1(1, 0, 0, 0)$  is locally asymptotic stable. This illustration is shown in Figure 1a. After it, we consider parameters for system (2) as Row No. *ii* of Table 1 and Row No. *i* of Table 2, then the conditions of Theorem 2 are satisfied and consequently  $E_2(0.8, 0, 0.2, 0)$  is locally asymptotic stable. The behavior of  $s, i, x, y$  with  $t$  is depicted in Figure 1b. Then, we take parameters and initial condition for system (2) as Row No. *ii* in Table 1 and Row No. *i* Table 2, then the conditions of theorem 2 are held, and so  $E_3(0.25, 0.5357, 0, 0)$  is locally asymptotic stable. The behavior of  $s, i, x, y$  with  $t$  is depicted. This description is plotted in Figure 1c. In the next step, we take parameters and initial condition for system (2) as Row No. *iv* in Table 1 and Row No. *ii* of Table 2.

Therefore, the conditions of Theorem 2 are satisfied, and so  $E_4(0.4884, 0.2519, 0.1589, 0)$  is locally asymptotic stable. This locally behavior is shown in Figure 1d. Then, If we take the parameters and initial condition for system (2) as Row No. *v* in Table 1 and Row No. *iii* of Table 2, then the conditions of Theorem 2 are satisfied and therefore the equilibrium point  $E_5(0.75, 0, 0.25, 0.4583)$  is locally asymptotic stable. This kind of stability is plotted in Figure 1e. Finally, we consider parameters as Row No. *vi* in Table 1 and Row No. *iv* of Table 2. Then the equilibrium point  $E_5(0.75, 0, 0.25, 0.4583)$  is locally asymptotically stable and  $s, i, x, y$  approach

to  $(s^*, i^*, x^*, y^*) = (0.2568, 0.1921, 0.1550, 0.1713)$ , which are shown in Figure 1f.

Now, we can solve our optimal control problem by the help of an iterative method numerically. We here obtain the optimality system from the state and adjoint equations. The optimal control problem strategy is obtained by solving the optimal system, which consists of eight ordinary differential equations and boundary conditions. By the Runge Kutta fourth scheme, we can solve the optimality system; see [4]. Starting with an initial guess for the adjoint variables, the state equations are solved by a forward Runge–Kutta fourth scheme in time. Thus, by a backward Runge Kutta fourth scheme, those state values are used to solve the adjoint equations because of the transversally conditions; see [13]. We present the results using the "bvp4c" and "bvp5c" subroutines in MATLAB software. To control in all infected prey and predator individuals, we use two control factors as treatment. We consider the treatment for 30 days, because a long treatment in the form of medication has a potentially harmful side effect and the best time of vaccination is the possible early stage of diseases. We here use a set of parameters value  $a, b, c, d, e, f, g, m, n, \alpha, \delta$  to determine the numerical simulation of the optimality system with a sufficiently small time step size. In Figures 2–7, we plot all population sizes in two systems having control and without control. The solid line denotes the population of individuals in the system without control while the dash-dotted line denotes the individuals population in the system with control. The changes of population is clearly visible for equilibria  $E_3$ ,  $E_4$ , and  $E^*$  in Figures 4, 5, and 7. The population of infected individuals,  $i(t)$  and  $y(t)$  is reducing during the time period, in these cases. Moreover, the population of the other susceptible individuals,  $s(t)$  and  $x(t)$  is increasing.

The equilibria  $E_1$  and  $E_2$  are not infected, then the control factors have no important effect on results as we see in Figures 2 and 3. In the other hand, after applying the control function  $E_3$ ,  $s_3(t)$  increases significantly and  $i(t)$  decreases. Figure 3 demonstrates the increase of prey and decrease of infection in prey population. Also, it reveals less decrease in predator population. After applying controls, Figure 7 at the  $E^*$  equilibrium shows the decrease at infected prey and predator, while increasing the predator populations.

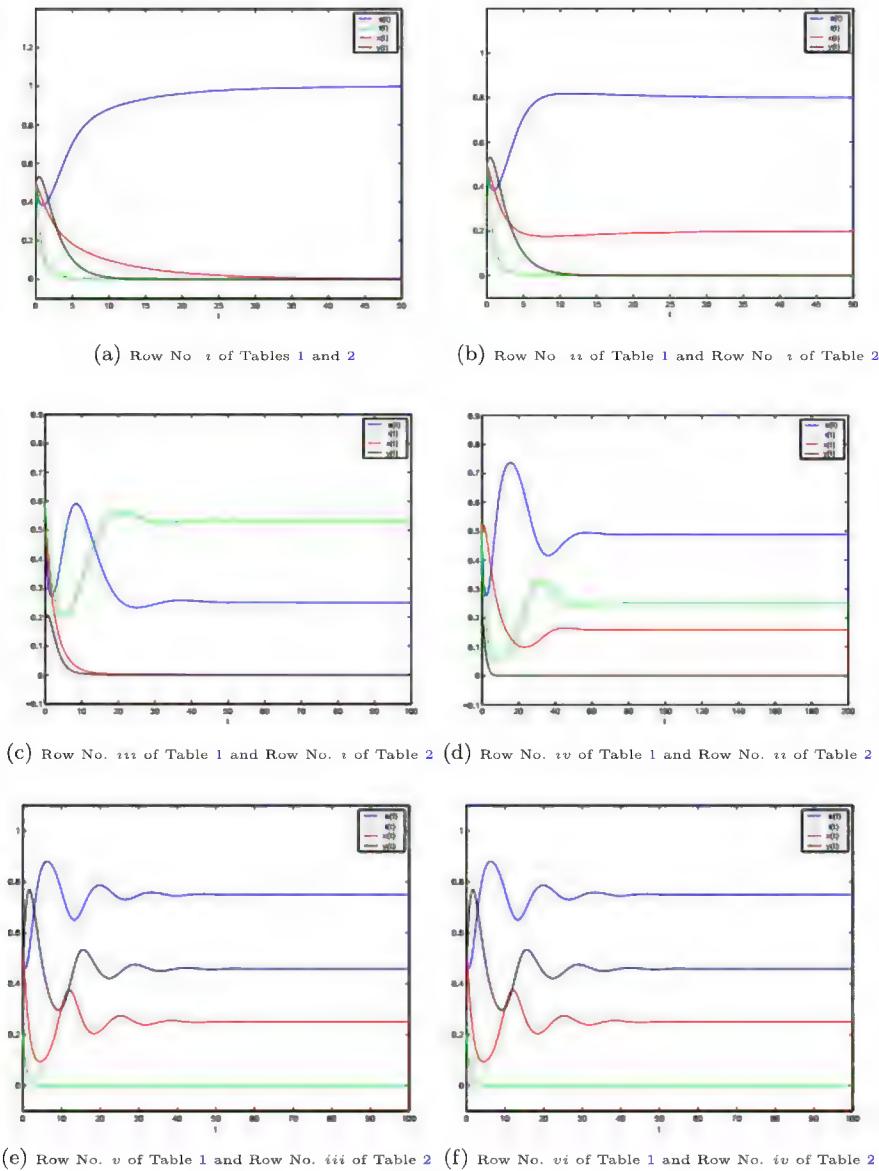


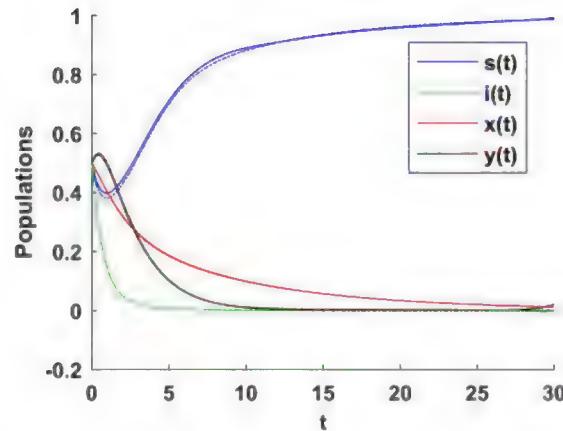
Figure 1: Stability of system (2) for different parameters and initial conditions.

Table 1: Parameter values for system (2)

Row No. Parameter	$a$	$b$	$c$	$d$	$e$	$f$	$g$	$m$	$n$	$\alpha$	$\delta$
i	0.4	1	0.1	0.5	0.2	0.2	0.15	1.5	1.4	0.5	0.2
ii	0.4	1	0.5	0.5	0.2	0.2	0.15	1.5	1.4	0.5	0.4
iii	0.4	1	0.1	0.1	0.2	0.2	0.15	1.5	1.4	0.8	0.4
iv	0.4	1	0.1	0.1	0.2	0.6	0.6	1.5	1.4	1	0.2
v	0.4	1	1	0.1	1.2	0.6	0.4	1.5	1.4	0.3	0.2
vi	1.9	1.2	0.8	0.2	0.2	0.2	0.15	1.5	1.4	0.3	0.2

Table 2: Initial conditions for system (2)

Row No. Initial value	$s(0)$	$i(0)$	$x(0)$	$y(0)$
i	0.5	0.5	0.5	0.5
ii	0.5	0.5	0.5	0.2
iii	0.5	0.2	0.5	0.2
iv	0.5	0.2	0.5	0.5

Figure 2: This plot shows the population of  $s(t)$ ,  $i(t)$ ,  $x(t)$ ,  $y(t)$  for equilibrium  $E_1$  with control (solid line) and without control (dash-dotted line).

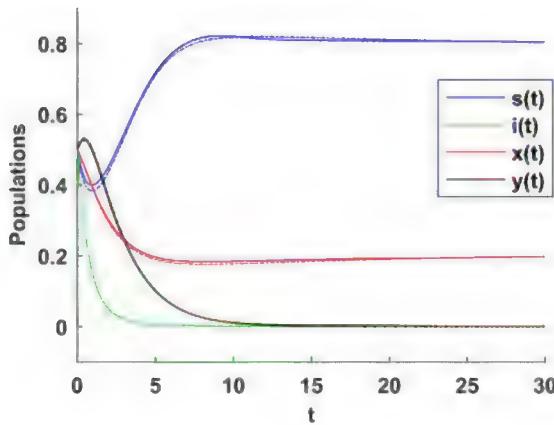


Figure 3: This plot shows the population of  $s(t), i(t), x(t), y(t)$  for equilibrium  $E_2$  with control (solid line) and without control (dash-dotted line).

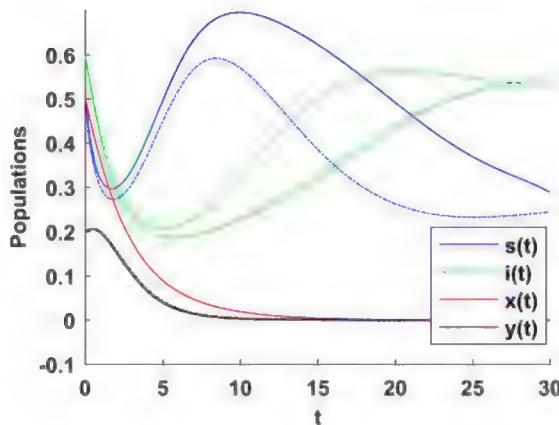


Figure 4: This plot shows the population of  $s(t), i(t), x(t), y(t)$  for equilibrium  $E_3$  with control (solid line) and without control (dash-dotted line).

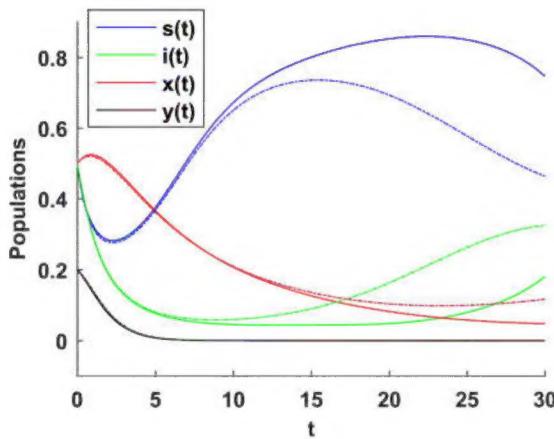


Figure 5: This plot shows the population of  $s(t)$ ,  $i(t)$ ,  $x(t)$ ,  $y(t)$  for equilibrium  $E_4$  with control (solid line) and without control (dash-dotted line).

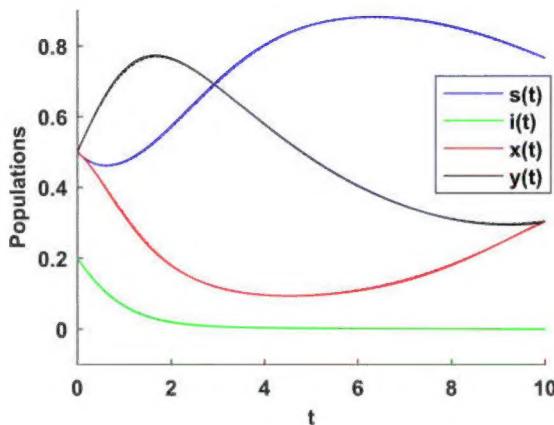


Figure 6: This plot shows the population of  $s(t)$ ,  $i(t)$ ,  $x(t)$ ,  $y(t)$  for equilibrium  $E_5$  with control (solid line) and without control (dash-dotted line).

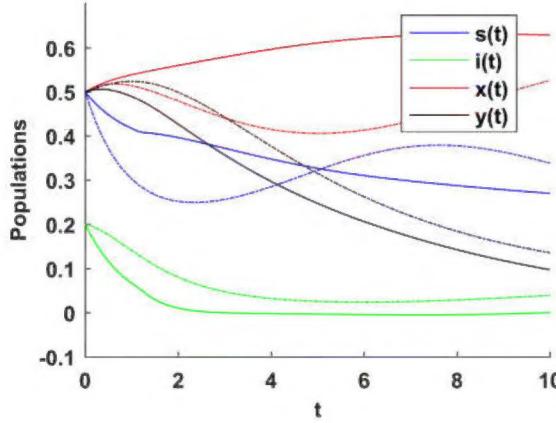


Figure 7: This plot shows the population of  $s(t)$ ,  $i(t)$ ,  $x(t)$ ,  $y(t)$  for equilibrium  $E^*$  with control (solid line) and without control (dash-dotted line).

## 6 Conclusion

Last century was known for mathematical physics era, but the present century belongs to biological mathematics, which has achieved considerably advanced developments in all branches of bioscience, medical, ecology, and so on. In the present research work, a four-dimensional Eco-epidemiological model having infection in the present species is studied. In other words, we formulated a prey-predator model having a disease in both of the populations. For almost all the models with diseased prey, we considered the predators live only on the infected preys (as they are weak and more vulnerable), therefore, the susceptible preys are completely out of danger is an oversimplification. In our model, we made more realistic assumptions which are as follows:

- (i) The susceptible predators are capable of catching both the susceptible and infected preys, and
- (ii) infected predators (being weak with disturbed internal mechanism) can manage only the infected preys (due to the same reasons).

The model may be used in many ways. Precisely, the behavior of solutions and equilibria such as existence, boundedness, and stability are investigated. Meanwhile the basic reproduction number for the proposed model is computed, the related control model and an optimal treatment are carried out. New controlled model are developed from the numerical simulation of the optimal system, which represents dynamics in each individual of the community. The controls developed in this paper support that the number of the susceptible and infected individuals decreases and the number of the recovered individuals increases in the optimal system. We also pointed out that

for certain values of the control rate, there exists its corresponding optimal solution. The above discussion is verified by the numerical simulation.

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